Effect of Valproic Acid on Proliferation and Apoptosis of Hepatocellular Carcinoma HepG 2 Cell Line

Masumeh Sanaei, Fraidoon Kavoosi, Shekoufeh Atashpour, Farshid Abbasi

Abstract

Eucaryotic cells genomes are organized in chromatin whose basic repeating unit is the nucleosome which consists of 147 base pairs of DNA wrapped 1.7 times around an octamer of histone proteins (two each of histones H2A, H2B, H3, and H4). Histone modification such as histone acetylation affects chromatin structure and gene expression. In fact, acetylation of lysines is regulated by the opposing action of two families of enzymes containing histone acetyltransferases (HATs) and histone deacetylases (HDACs) that together determine the acetylation status of histones. The balance of histone acetylation and deacetylation plays a critical role in the regulation of gene expression. Aberrant activity of HDACs, however, leads to tumorigenesis. Histone deacetylase inhibitors (HDACIs) are a novel class of chemotherapeutic agents that target the classical HDAC enzymes by which activate differentiation programs, inhibit the cell cycle, and induce apoptosis. HDAC inhibitor valproic acid (VPA) has antitumor activities against certain cancers. The aim of the present study was to analyze the effect of VPA on proliferation and apoptosis of hepatocellular carcinoma HepG 2 cell line. Materials and Methods: MTT assay and flow cytometry assay were used to evaluate proliferative and apoptotic effects of VPA. Results: VPA inhibited the growth of HepG 2 cell and induced apoptosis significantly with a time- and dose-dependent manner. Discussion: Our finding clearly indicated that VPA has a significant inhibitory and apoptotic effects. Conclusion: VPA can significantly inhibit the growth and induce apoptosis in the HepG 2 cell line.

Keywords: Valproic Acid, Proliferation, Apoptosis, Hepatocellular